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There's an elephant in the room: The impact of early poverty and neglect on intelligence and common learning disorders in children, adolescents, and their parents

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Recognition of poverty and neglect is very important in formulating a practical diagnosis of children's and adolescent's learning disorders. Early brain development forms the basis of learning, behavior and health over the entire life span. Through accumulated stressors and lack of supports, early poverty undermines competent parenting which then impairs learning and is the most common cause of mild mental retardation (MMR). Parenting interventions left until school entry do not prevent the insidious decline in IQ over the developmental period. Most types of learning disorders (e.g., ADHD, LD, FAS/FAE) are either aggravated or actually caused in the presence of chronic early poverty. Lack of functional accessibility to family, extra-familial and community resources for the poor impairs their adjustment to their children's learning disorders. Stresses due to poverty and cultural marginalization also exacerbate FAS/FAE. Poverty and despair eclipse diagnostic debates in producing hyperactivity, memory impairment, and social maladjustment. The need for early identification of at-risk infants and the provision of nurturing stimulation, cognitive training and parental support cannot be over-emphasized.

Treating children with learning disorders is a significant challenge. A complete and accurate history is therefore crucial. The early years are very important. Issues of poverty and neglect are just as important currently (Evans, 2004; Garber & Begab, 1988; McCain & Mustard, 1999) as they were in previous decades (White, 1975). Early neglect impairs brain growth and development (Haydar, 2005) and produces mild mental retardation (*MMR*), but seldom is the effect of poverty and neglect integrated with our understanding of the etiology of prominent specific learning disorders such as learning disabilities (*LD*s), attention deficit hyperactivity disorder (*ADHD*) and fetal alcohol syndrome and

effects (*FAS/FAE*). This is a serious oversight, as a close inspection of the literature reveals that poverty is often the most important variable in understanding children's learning disorders. Early poverty sets the stage for neglectful parenting in which children lack suitable stimulation and support. The current review identifies the special threats that poverty poses for children's cognitive development generally and for common learning disorders in particular.

As we illustrate, when children and adolescents suffer from *MMR* either alone or accompanied by other learning disorders, the quality of early intervention is suspect. As we illustrate, financially and socially stable families are significantly more able to adequately cope with their children's special needs for nurturance and remedial help. Such offspring then fare much better as adults. As mental health specialists and educators, poverty is an integral part of our business as it has a huge impact on behavior. As our practices become more interdisciplinary, economic and cultural factors will figure more prominently in assessment and treatment. As we illustrate, severe early poverty diminishes adequately adaptation to life's challenges, including those that accompany cognitively-based developmental disorders (Pennington, 1991).

Living contexts (e.g., Reiss, Collins, & Berscheid, 2000) such as early poverty and the pressures that they bring are quite relevant to developmental learning disorders. Evans (2004) provides a stellar review of the damaging multidimensional and intergenerational effects of poverty on young children.

While the quality of subsequent years is also important (Kagan, 1996; Reynolds, Ou, & Topitzes, 2004; Thompson & Nelson, 2001), the early years are crucial. Competent parenting in the early years provides an optimal protective shield against subsequent educational decline, economic disadvantage, mental illness and involvement in crime. As Garber and Begab (1988) and White (1975) claimed, the impact of the preschool experience is quite telling. For instance, Feinstein and Brunner (2004) found that children who eventually escaped from the bottom *SES*

quartile developed similarly to those who remained because their early years had such a profound and enduring impact on them.

It is important to observe the child's micro-environment and parenting behaviors in particular when understanding the dynamics of poverty and neglect. Neglectful parenting is the antithesis of competent parenting. On the other hand, enhanced parental competence is intricately connected to the special demands, such as attentiveness, patience, and control, inherently manifested in dealing with both normal and especially disabled children. These extra demands are stressful to any parent but for the vulnerable also serve to degrade adequate parenting and accentuate the severity of the child's behavior. Such bi-directionality works to undermine effective parenting and places children at greater risk. For example, even otherwise competent parents are often reduced to excessively harsh and inept behaviors when placed in a position of interacting with conduct disordered (*CD*) children (e.g., Anderson, Lytton, & Romney, 1986) and adolescents (e.g., Rhule, McMahon, & Spieker, 2004), or *ADHD* children (e.g., Smith, 2000). These challenges on poverty stricken and neglectful parents are much more serious.

Hyperactivity is a common co-morbid feature in children's and adolescents' learning disabilities (Pennington, 1991) which stress parents and teachers alike (O'Connor, 1995). The net result is a coercive cycle of harshness or neglect, wherein overburdened parents may place their immediate emotional needs above those of their demanding offspring. This is especially true of poverty stricken parents who so often lack sufficient intellectual and emotional resources to overcome their children's adverse behaviors. In contrast, middle income parents of such children are much more likely to cope better by taking advantage of useful school contacts and community supports (e.g., Evans, 2004). As Evans indicated, families which adapt well to their developmentally disabled children have relatively strong marital relationships, deal well with their children's specific characteristics (e.g., mild communication impairment), have the availability of parental support groups, small intense social support networks, higher *SES*, fewer offspring and more supportive communities (Kwai-sang Yau, Cecilia, & Li-Tsang, 1999).

Fractured, socially unsupported, isolated and poverty stricken single parent families are at higher risk of adjusting poorly to their children, are more apt to aggravate a level of cognitive impairment and are more likely to actually foster cognitive disabilities in subsequent offspring.

Mild Mental Retardation (MMR)

We focused on *MMR* because it is primarily environmentally generated by poverty and neglect and is the most common sub-type (e.g., Bradley, Thompson, & Bryson, 2002), accounting for approximately 3.54/1000 of Canadian adolescents. On the other hand, *MR* includes about 1-3% of the industrialized population. There are more than 1000 known causes (Dykens, 2003) of *MR*. For example, Down syndrome (*DS*) and Fragile X Syndrome (*FXS*) are the two most frequent genetically caused types of *MR* and appear more independently of parental circumstances such as poverty and neglect. For example, a diagnosis of *DS* is not significantly related to *SES*, sex, maternal age or race (Neser, Molteno, & Knight, 1989; Tyler, Synder, & Zyzanski, 2001). Such children are also more likely to encounter physical problems as they age, such as osteoporosis (Centre, Beange, & McElduff, 1998) and cardio-vascular problems related to elevated body mass index (*BMI*) (e.g., Gibson, 1997). Moreover, interventions are far less successful in achieving lasting improvements (e.g., Tyler et al.). Also, Neser et al. found that children with *DS* decreased in developmental quotient (*DQ*) with age, regardless of their *SES*. Such is not the case with *MMR* (Edgerton, 1984; Garber & Begab, 1988), which is very *SES* dependent in the early years (Garber & Begab, 1988). However, in contrast to their more genetically caused counterparts, such children respond very well to early intervention.

Normal development is precisely what is threatened either prenatally by a variety of genetic disorders or teratogens (e.g., Haydar, 2005) or postnatally by early poverty and neglect (e.g., Garber & Begab, 1988). Lack of early stimulation at an adequate level interferes with brain development (Shaw et al., 2006). By definition, *MMR* is at least two standard deviations (i.e., 30-32, depending on the tests used) below normal and most (75-85%) types of intellectual delay are mild or "cultural-familial" (*IQs* >50<70-75) (Garber & Begab, 1988; Valente, 1989)

and are usually accompanied by adaptive difficulties. On average, competent parenting begins to be seriously threatened even when *IQ* is within the borderline level (*IQ*s >70-75 <80) (Garber & Begab).

The importance of the early years is stressed by Sapolsky (2005) who found that lower *SES* is related to poorer health outcomes. Of particular interest is that American nuns sharing similar current lifestyles had similar patterns of disease and dementia related to their *SES* status 50 years earlier. Controlling for risky lifestyle behaviors and protective ones, such as access to medical care, apparently accounted for only about one third of the illness difference. Living in early poverty, with its attendant chronic stresses and immuno-suppression, accounted for the rest of the variance. The deleterious effects of chronic stress and sustained gluco-cortisol release on neurological health in well known (Feldman, Meyer, & Quenzer, 1997). Cortisol is damaging to nerve cells, particularly those involved in memory and new learning. Thus, dealing with poverty and preventing its deleterious long-term effects is of paramount concern. Regardless of race and culture, lower working class (*LWC*) parents of *MMR* children themselves scored lower on adaptive behavior than did their lower middle class (*LMC*) counterparts (Slate, 1983). Thus, addressing the health and stability of the family within a supportive community is integral to a competent assessment and intervention of children at risk and of children with cognitive disabilities in particular.

Early childhood vulnerability and MMR

McCain and Mustard (1999) produced one of the most ambitious works dealing with the early years, finding that they are crucial for laying the groundwork for child outcomes. Literature gleaned from the neurosciences, developmental psychology, the social sciences and other relevant disciplines, confirmed White's (1975) earlier claims. Adequately supportive and stimulating early years forge the intergenerational quality of intellectual and interpersonal competence. McCain and Mustard's position is that brain development, within the first six years of life, forms the basis of learning throughout the lifespan.

The *Milwaukee Project* (Garber & Begab, 1988) also found that negative early experiences, such as severe neglect and lack of appropriate verbal stimulation, are likely to have profound adverse long-term effects on intellectual development. As with McCain and Mustard's (1999) report, adverse early negative experiences, such as socio-economic privation, influenced subsequent learning, behavioral and health problems. Parents in such households were significantly less likely to provide their children with the linguistic and quantitative training they needed. Adaptive behavior and poverty are often closely connected (e.g., Ackerman, Brown, & Izard, 2004; Mistry, Biesanz, Taylor, Burchinal, & Cox, 2004). Garber and Begab also found that *MMR* was almost always associated with adverse parenting environments accompanying sustained poverty. Unfortunately, by the time *MMR* is identified in the schools, it is fairly resistant to change.

Using a *social deprivation hypothesis*, Garber and Begab (1988) provided compensatory preschool interventions for the mothers of at-risk preschoolers. This middle-class micro-parenting environment stressed language and teaching strategies which were provided to the at-risk mothers and supplemented by on-the-job training and counselling and help with household tasks. (Within the at-risk group [n = 88], 45.4% of the mothers with *IQ*s <80 accounted for 78% of children with *IQ*s <80. These percentages increased with even lower maternal *IQ*s.) Without intervention, children of such mothers had a progressive decline in *IQ* to the borderline level by age 14. Intervention included enrollment in the *Infant Stimulation Centre,* involved short visits for five days a week, and began at seven hours per day. Maternal rehabilitation in reading, writing and math occurred for four days, over four weeks. Child tasks involved school readiness and instruction by qualified teachers. Parental interventions stressed imitation, memory, vocabulary, concept formation, predictive labeling, listening skills, speaking, demonstrating, helping and conversing.

The results were noteworthy. By 22 months, the intervention children were at least as comparable to the low-risk contrast children in most key respects on motor, adaptive, personal-social and performance tasks. However, they appeared to be superior in measured language skills.

These comparisons widened over time at 72 months and persisted and grew by age 10. Later interventions done *post hoc* strongly suggested that such attempts were relatively ineffective at arresting the insidious intellectual decline of the at-risk contrast group.

Evans (2004) carefully documented the cumulative harmful effects of poverty, which include negative, harsh and unresponsive parenting, exposure to aggressive peers, family instability and divorce, lack of parental monitoring, lack of maternal emotional support, weaker social ties, less cognitive stimulation, less parental involvement in school activities, residential instability, excessively crowded and noisy living arrangements, less nutrition and greater exposure to environmental toxins, such as lead, tobacco and alcohol. Sameroff, Seifer, Baldwin, and Baldwin (1993) also found that, as the number of comparable risks increased, children's *IQ*s decreased. Such risks included poor maternal mental health, high maternal anxiety, low maternal education, head of household unemployed or unskilled, father absent, minority or group membership, more than four children in the household and a high incidence of stressful events such as job loss or death in the family.

Socio-economically quantitative approaches to poverty (e.g., Ackerman et al., 2004; Mistry et al., 2004) show that there may be an economic cut-off point below which chronically poor (i.e., at-risk, *Head Start*) children do markedly worse in terms of their readiness to learn and adapt to society and a cut-off point above which benefits asymptote. Poverty was defined using an income-to-needs ratio. When *per capita* poverty level fell below a ratio of 2, recent and persistent risk effects occurred more frequently. By grade 5, the list of adverse outcomes associated with poverty was lengthy: externalizing and internalizing behavior problems, lower academic performance, relationship instability with externalizing problems, academic incompetence, and police contacts. Internalizing problems coincided more with psychiatric morbidity. Protective factors were children's cognitive ability and maternal schooling, which correlated with the children's academic competence. However, harsh parenting was negatively related to academic competence.

For non-identified at-risk children, the effects of poverty are rather more cumulative. Using 1,364 preschoolers selected from 10 hospitals, Mistry et al. (2004) found that the income-to-needs ratio was linearly related to better family processes and child outcomes. However, as with Ackerman et al. (2004), beyond an income-to-needs ratio of 5, benefits seemed to asymptote. At the poverty level, family processes such as perceived financial maternal depression, maternal insensitivity, lack of appropriate child cognitive and language development, and maternal reports of child behavioral and social problems fully accounted for the income effects over this period of time. Poverty itself is not necessarily the culprit as inattentiveness and child behavioral problems have also been linked to lack of parental involvement in normal populations (Baumrind, 1967; 1971; 1991).

As with Evans (2004), Sameroff et al. (1993), and Mistry et al. (2004), Burchinal, Roberts, Hooper, and Zeisel (2000) found that such early risk factors as maternal relationship instability, police contacts, psychiatric morbidity, harsh parenting, and low maternal schooling were cumulative in their deleterious effects on young children. Risks, such as lack of maternal education, household size, lack of maternal involvement (Harms & Clifford, 1980; Harms, Clifford, & Cryer, 1998; Harms, Cryer, & Clifford, 1990; Marfo, 1992), poor child-rearing environment (Elardo & Bradley, 1981) and poor quality centre-based care (Harms & Clifford) were significantly correlated with poorer child outcomes by 36 months; thus, especially for at-risk populations, where there is a history of stressful levels of chronic poverty and maternal dysfunction.

Adaptive behavior and MMR

The connection between intellectual ability and adaptive behaviour, including socially adaptive behaviour, is stronger for milder forms of *MR*. For example, Kicklighter, Bailey, and Richmond (1980) examined the relationship between academic status and social competence in a group of 7-10 year-olds. Compared to the (i.e., borderline *IQs* >70-75<80) slow learners (*SLs*), the authors found that the overall correlation was .51, with the *MMR* children scoring lower on all adaptive domains.

Lack of success with one's peers is very important in terms of negative life events, such as delinquency (e.g., Bigelow, 2000). Lack of social integration is perhaps the most serious adverse effect of most any disability or disorder as it alienates the person from valuable and necessary forms of information and self-validation. A host of occupational, educational, and personal adaptations are predicated upon competent social skills acquired chiefly through the peer group. Indeed, socially rejected children are at high risk for acquiring anti-social peer influences. Happily, early interventions with parents of inner-city preschoolers have been linked with reductions in delinquency - a high risk for multi-stressed families (Miller, 1994) - as well as significant reductions in mild mental deficiency (Garber & Begab, 1988). Leffert, Siperstein, and Millikan (2000) found that children with *MR* resembled younger children without *MR* in terms of their ability to correctly interpret another child's intentions (i.e., as "being mean"). Consistent with the work of Dodge (e.g., Crick & Dodge, 1994), socially maladjusted normal *IQ* children often have inaccurate perceptions of other children's intentions, misperceiving untoward motives and acting antisocially. In other words, such children are more apt to interpret entirely neutral occurrences (e.g., accidentally bumping into another person) as aggressive acts and to retaliate. Given the inattentiveness, children with *MMR* are more at risk.

Delaying interventions for socially maladaptive behavior is a problem. Santich and Kavanaugh (1997) found that partial school integration of 32 *MMR children* from grades 3-6 had negative consequences for them in terms of lack of social acceptance, stemming from higher levels of their inappropriate behaviour. These delayed students were also less likely to be preferred for play or selected as best friends. In fact, Taylor, Asher, and Williams (1987) found that, compared to controls, *MMR* children were generally rejected by their peers. They were perceived as more shy and avoidant, less cooperative and less leadership-like. One subgroup was aggressive. Of keen interest, social rejection was not confined to the peer group, as Leyser and Abrams (1982) found that teachers as well were least accepting on a social distance scale of those students who were mentally retarded, emotionally distant or delinquent, compared to those who were normal or gifted. Students who were perceptually and

physically handicapped fell in-between. Social skills training, such as involvement in *Special Olympics* (Dykens & Cohen, 1996), is encouraging in this regard. Indeed, Harter (1990) has found that the peer group is a main source for self-esteem in the elementary school years and that self-esteem (e.g., sports, academics, etc.) is domain specific.

Attention Deficit Disorders and Poverty

Aside from *MMR*, the single most common behavioral and learning problem, in North America at least, is Attention Deficit Hyperactivity Disorder (*ADHD*), occurring in 3-7% of school-age children. This disorder is developmental in origin, usually identified by age 7 and is often first detected when the child must function within the strictures of a school setting. Often school work begins to suffer as seat work becomes too difficult to sustain. Classifications may be of the inattentive (*ADD*), hyperactive (*ADHD*) or combined types (Barkley, DuPaul, & McMurray, 1990; Barkley, Edwards and Robin, 1999; Conner, 1990) and often also includes an additional diagnosis of Oppositional Defiant Disorder (*ODD*) and/or Conduct Disorder (*CD*) (Pennington, 1991). Interestingly, McCauley, Feuillan, and Ross (2001) found that Tourette's syndrome and Turner's syndrome (*TS*) are often also co-morbid with attention disorders.

Galili-Weisstub and Segman (2003) flagged *ADHD* as a common idiopathic childhood neurodevelopmental disorder, which impairs schooling and social adaptation and is often accompanied by depression, *CD*, early school leaving and substance abuse proneness. As we show below, hyperactivity is also a key aspect of *FAS*/*FAE*, a common cause of *MR*, and can confuse a valid *ADHD* diagnosis. *ADHD* disorder has a known heritability involving dopaminergic (DRD4) genes, which is likely the reason that many *ADHD* adolescents, like their adult counterparts, present with smoking and drinking problems as well. Other neurotransmitter systems (e.g., seratonergic, noradrenergic, cholinergic) may also be implicated.

Adapting to ADD/ADHD

The behaviors of children with developmental disorders, such as *ADHD*, can be very demanding to parents and teachers alike. Rosenzweig, Brennan, and Ogilvie (2002) conducted a focus group study in which they found that normal parents usually adjusted reasonably well to their offsprings' difficulties by adapting their work responsibilities and day care arrangements to make a better fit between their own and their children's needs. Work adjustment also involved taking jobs which were less demanding and which required fewer hours of work. Interestingly, none of these caregivers placed their children in a day care or an after school centre; they utilized their own social networks. It was usually the case that the affected children were cared for within the home by siblings, the parent, or an adult specifically hired for the purpose. It is also worth noting that these parents were not parentally challenged and economically deprived.

Rosenzweig et al. (2002) noted that lack of knowledge of the school personnel and the parents' work supervisors were key sources of frustration for such parents. Evidently, these parental perceptions of lack of school support were not without a factual foundation. Matlock (2000) found that teachers of *ADHD* elementary school children who were specifically trained in appropriate teaching strategies and interventions were more helpful. Thus, competent parents act as advocates for their children, martialling familial and extra-familial supports and are keenly sensitive to expertise.

How families cope with their children's *ADHD* and other developmental disabilities is pivotal in helping to prevent parental depression and burnout (Herman & Marcenko, 1997) and is essential in planning any suitable intervention. In a qualitative interview study of 17 families with children who have *ADHD*, Segal (1998) found that normal families changed their routines to different scripts in order to foster their children's occupational success. These parents used strategies such as making a game of getting dressed, and delaying their own grooming until the evening period. Segal (2000) found that mothers of *ADHD* children often coped by multi-tasking (i.e., temporal enfolding) and by

chunking or delegating work. Holderness (1998) observed 25 pre-adolescent boys diagnosed with *ADHD* and followed up 18 of them five years later. It is important to note that these boys did not have *CD*, *ODD*, or other co-morbid disorders complicating the picture. Compared to normative families, these target families were highly adaptive, but were understandably also more highly stressed over time. As one might well expect, family cohesion, communication and good problem solving skills were related to lower levels of problem behaviors and higher self-concept scores. What was not known was whether higher family cohesion stemmed from less disruptive forms of *ADHD* behaviour, but one can generally expect this to be the case (Anderson et al., 1986). Given the importance of family cohesion and adaptive behavior in coping with their offsprings' *ADHD*, the implications of parents having *MMR* and living in poverty are quite onerous.

Co-morbidity with mental illness

The overlap of *ADHD* with mood disorders and depression in particular, is as high as 30% (Schmidt, 2002). Schmidt found that when children and adolescents from ages 7 to 17 where evaluated, along with reports from their parents and teachers, the comorbid group had no greater degree of automatic depressive thoughts than did the depressed group. However, the comorbid group had more negative views about the world, the future and a more negative attributional style. Also, Carter et al. (2000) found that children and adolescents who suffered from Gilles de la Tourette's syndrome had significantly more internalizing, externalizing and social problems if they also had comorbid *ADHD*. Thus, when in the context of *ADHD*, depression, behavioural and social problems are significantly more entrenched. This connection of behavioural difficulties with *ADHD* was also evidenced in a Quebec five-year high school longitudinal study. Royer, Couture, Fortin, Potvin, and Marcotte (2000) found that the hyperactive *ADD* school children were significantly more depressed, had lower achievement scores and had more disciplinary and social problems, including higher rates of school absenteeism. It is thus very unfortunate that parents of *ADHD* children often feel that they cannot rely on extra-family supports (Rosenzweig et al., 2002), as Frame (2002) found that preadolescent support groups over

eight sessions significantly enhanced participants' feelings of social acceptance, physical appearance, athletic competence, and global self-worth. Such therapeutic improvements are bound to reduce parental stresses as well.

Etiology of ADHD: abuse and neglect. It appears that *ADHD* has many potential contributors; that is, the principle of *equifinality* is alive and well. The etiology of *ADHD* has a known familial-genetic linkage (e.g., Galili-Weisstub & Segman, 2003) but there is also an interesting connection between physical and sexual abuse and neglect. While Garber and Begab (1988) found clear evidence that maternal neglect is causally linked to children's longer-term intellectual and social impairment, the linkage to hyperactivity is also intriguing. In a prospective study, Sexton (1999) found that abuse and neglect are linked to a variety of mood, learning and conduct problems, such as depression, thought disorders, *CD* and *ADHD*. Moreover, Sexton found that abuse coupled with neglect had the most deleterious outcomes. One must also be mindful of the increased stresses that such disorders have on parents who are also having difficulties coping. It is also remiss not to address an etiology of *ADHD* with maternal use of ethyl alcohol. That is, a disorder, such as *ADHD*, or one presenting as *ADHD*, may have a number of different contributors, but alcohol is potentially one of them, thus confounding an accurate diagnosis. Given the fact that *ADHD* and *FAS/FAE* share core symptoms such as impulsiveness, inattention and hyperactivity does not necessarily mean that treatments will be equally effective. Pennington (1991) stressed that *ADHD* may be a primary disorder or secondary to other learning disorders, such as *LD*. The reverse may also hold. Thus, poverty, with its high level of family disruption, conspires to increase the risk of not only intellectual delay, but also of *ADHD* symptoms through lack of competent parenting and neglect and relatively lower usage of community and family and extra-family supports.

Learning Disabilities

We operationally defined learning disabilities (*LDs*) as a significant problem in academic underachievement – usually of at least two

academic years – which is not primarily due to sense organ impairment, such as deafness or blindness. An *LD* is not primarily associated with an emotional disorder unrelated to the impairment itself, and is not a product of general intellectual disability or mental retardation. At least one author (Daniel, 1996) views *LD*s as essentially language-based neurological disorders. Bannatyne (1968; 1974) stressed the difficulties in informational sequencing and organization of *LD*s. The prevailing notion is that *LDs* are either verbal or nonverbal organizational informational deficits of comprehension (i.e., receptive) or production (i.e., expressive) in educational, employment and social settings (Weller, Watteyne, Herbert, & Crelly, 1994). Common *LDs* may involve problems in understanding (i.e., receptive aphasia), expressing (i.e., expressive aphasia), an inability to read (i.e., dyslexia), or specifically in terms of mathematical ability (i.e., dyscalculia).

Layton and Lock (2001) also addressed the issue of dual diagnoses in *LD* children. As with other learning and behavioural disorders, dealing with comorbid conditions is essential. To illustrate, McGrother, Hauck, Bhaumik, and Thorp (1996) measured 2,117 *LD* adults, finding that behavioural and psychological problems, including epilepsy, were common concerns. It should also be made clear that an *LD* is not simply academic underachievement due to lack of sufficient training. Many students have learning problems or educational underachievement and do not have an *LD*. These students need effective compensatory education. Clues here are academically unsupportive parents and long-standing attendance problems, coupled with a normal *IQ* and few or no problems with solving basic intellectual problems not requiring specialized instruction.

As with *ADHD*, learning disabled children often have social adjustment problems. Gadeyne, Ghesquiere, and Onghena (2004) examined *LD* in first-graders, defining specific *LDs* in terms of achievement falling 1.65 standard deviations or more below *IQ* expectations and whose math or reading scores were significantly discrepant from each other. Along the same lines as O'Brien (2003), learning disabled children were far more likely than their low achieving counterparts to have deficiencies in attention, total behaviour and total problems recorded on the *CBCL*.

Learning disabled children were different from those who were simply low achievers in that the low achievers tended to exhibit lower self-concepts, whereas the *LD* children were more apt to have identifiable social difficulties. Watts (1986) portrayed a troubling tendency for LD adolescents to have more serious adjustment problems compared to their normal counterparts.

Adaptation and poverty. Adjusting to the diagnosis of *LD* is of course crucial to its remediation, especially as it is likely a life span problem (Smith, 1987). Sorensen et al. (2003) examined the psychosocial adjustment over a two-year period of 100 children with *LD*. Even though academic difficulties were understandably chronic, it was the contextual factors, such as family and school supports, that were most important in determining adaptation and adjustment that affected psychosocial outcome. Moreover, different factors were relatively more important for parents, teachers and children. Rigazio-DiGilio and Cramer-Benjamin (2000) found that the family's stress experienced as a consequence of the child's *LD* was a function of the child's age and the level of uncertainty of the diagnosis. An early and definitive diagnosis was related to lowered parental stress. Parental stress and poverty are indeed related to adaptation to the child's *LD*.

Reinforcing Rigazio-DiGilio and Cramer-Benjamin (2000), Morrison and Cosden (1997) noted that *LD* itself does not necessarily imply that the affected individual will necessarily have serious problems, such as poor emotional or family functioning, dropping out of school, delinquency, and abusing drugs. Indeed, it is the presence of the affected person's "internal" characteristics coupled with the characteristics of the family, school and community that serve to be protective. Interestingly, Werner (1993) investigated the development of 22 *LD* Kauai children from birth to age 32, and found that most of them had successful adaptations to adult life. While employment, marriage and divorce rates were comparable to age cohorts, mental health and criminal problems declined from adolescence to adulthood. Reminiscent of Garber and Begab (1988), in addition to temperamental, efficacy, planfulness, and self-esteem factors, resilience was related significantly to the availability

of supportive and competent adult care-givers. Such parenting is less common within chronic poverty.

Provocatively, McBurnett and Pfiffner (1991) argued that, particularly if it is moderate to severe, *ADHD* actually interferes with the psychological processes necessary for learning and is therefore a type of *LD*. Attention Deficit Hyperactivity Disorder may actually interfere with rule learning. In a clinical ABAB/BABA design, Bicard (2002) found that five *ADHD* children adapted well to the rules for learning math problems, but the rules had to be consistent and that this stability of contingencies may slow adaptation to the ebb and flow of the real world. It should come as no surprise, therefore, that rule-learning *LDs* are common in delinquent populations. Zinkus, Gottlieb, and Zinkus (1979) found that, of their 55 male delinquents, 73% were two or more grades below expectation in reading and 87% in spelling and arithmetic. In addition, about 80% of these subjects had auditory or visual-spatial processing deficits, such as errors on the *Bender-Gestalt* and mixed eye-hand dominance. These learning disabled children also had more difficulty acquiring grammatical morphological rules, such as the third person singular, possessives, and adjectival inflections. Moreover, Kravetz, Faust, Lipshitz, and Shalhav (1999) found that *LD* children in the 4th and 5th grades were significantly lower in interpersonal understanding and social adaptation in the classroom. Social understanding is normatively necessary in dealing with the world of peers (Bigelow, Tesson, & Lewko, 1996).

It should, therefore, not be at all uncommon to find that such *LD* children often adapt poorly to their peers (e.g., Santich & Kavanagh, 1997) and often have early onset *CD*. Hatzichristou and Hopf (1993) also noted such lack of social acceptance, as well as academic difficulties. Moreover, it should not be counter-intuitive to learn that such adolescents gravitate to imitating deviant behaviour with other delinquents. Indeed, this is precisely what a recent review (Bigelow, 2000) of the trajectory of delinquency found. Consistent with the commonly disruptive family origins of *LD, CD* and *ADHD* children, Sexton (1999) found that maltreated/neglected children are also at risk for developing relative underachievement and *LDs.* Even nutritional

deficiencies, such as in terms of iron and calcium, in fetuses and infants, may be associated with *LD* (Haas & Harrison, 1977). The *bi-directionality* issue is again very pertinent, as parents without a history of social support and effective models of parenting may be substantially more vulnerable to the cumulative effects of stress and far less able to nurture and protect their offspring. Adding to this behavioral brew, parental hyperactivity, impulsiveness and abuse may result. Such maladaptive parenting is frequently more common in poverty.

Those caring for these affected children and adolescents, particularly those who are informal carers, have financial, support and housing needs, including the need for respite care (McGrother et al., 1996). Interestingly, these informal carers had 40% more health concerns than the normative population and had four times the incidence of depression. Depression is a known risk factor for neglectful parenting, raising the spectre of child neglect and abuse amongst these cognitively disabled children, who are themselves inherently more challenging to these parents. While other disorders, such as autism, are even more stressful to care-givers (e.g., Wadden, 1995), given the importance of family supports in dealing effectively with *LDs (*Rigazio-DiGilio & Cramer-Benjamin, 2000), this study underscored the need to take better care of our care-givers. The impact of persistent poverty on adapting to a hyperactive and learning disabled adolescent can be expected to be quite substantial.

Aboriginal issues and cognitive disabilities: The poverty equation

Aboriginal peoples in North America are at particular risk for learning and behavioral disorders as a product of maternal alcohol use during pregnancy, as well as a product of being marginalized culturally, with the poverty and stresses that often accompany this cultural status. In fact, Rudmin (2003) reviewed the prevailing theories of acculturative stress and concluded that there is no convincing evidence that biculturalism is inherently adaptive, whether this is in terms of Natives, immigrants, refugees or sojourners. Thus, practitioners working with Aboriginals need to be very sensitive to acculturation stressors cautious

when making diagnoses of intellectual delay and other learning disorders.

As Allen (2002) claimed in terms of *American Indian* and *Alaskan* Native settings, test administration and the lack of local norms and the problems of adapting psychiatric diagnoses to local cultures can hamper valid interpretations. In a particularly spirited account, Darou (1992) described to his dismay how he discovered that a Canadian group of Native counsellors he was teaching had children who were tested with the *WISC-R*, in spite of lack of established validity of this test on aboriginal populations. Using examples taken from the *Otis-Lennon* (1967) test, depressed verbal scores may be an artifact of *Algonkian* language transfer errors and lack of item relevance. In contrast, Mueller, Mulcahy, Wilgosh, Watters, and Mancini (1986) found that the *WISC-R* was reasonably internally consistent for *Kitimeot* and *Keewatin* Natives of the *North West Territories*. But for these *Inuit* children, fully one third of test items fell within extreme ranges, with Verbal items proving too difficult and Performance items too easy, causing these researchers some interpretive and diagnostic concerns.

Salois (1999) found that, compared to the norm, *Northern Plains* Native American Indian children's performance on the *WISC-R* was significantly lower for Full Scale and Verbal IQ scores. While Performance Scale *IQ*s were not significantly different as a whole, visual-perceptual and visual-spatial-motoric abilities were particular strengths. In particular, Picture Completion and Mazes were significantly higher than the norm. Simultaneous as opposed to sequential reasoning was predominant. Congruent with this pattern, Wright, Taylor, and Ruggiero (1996) found that, when compared to White Quebec norms, Inuit Quebec children (ages 4-6) performed significantly better during the first two years of formal education on the Raven's Colored Progressive Matrices (*CPM*). Parenthetically, Garber and Begab (1988) in *The Milwaukee Project* found that puzzle solving – non-verbal problem solving like the *CPM* - was not sensitive to the effects of poverty. Wright et al. also found that *CPM* scores were not affected by the ethnic mix of the children's parents, whether they were both Native or of mixed Native/white heritages. The language of instruction and teachers'

ethnicity were also unimportant in affecting these scores. These authors concluded that academic underachievement is produced by not allowing these children to capitalize on their strengths in learning specific material. We disagree. Non-verbal measures of intelligence are least affected by poverty and neglect.

To illustrate, St. John, Krichev, and Bauman (1976) found that *WISC* Performance *IQs* were within the published norms for *Cree* and *Ojibwa* children but their Verbal *IQs* were in the deficient or dull-normal range. Also, Atkinson (1995) found that Native American children who were referred for speech and language or general purposes were significantly lower on the *Wechsler Individual Achievement Test* (*WIAT*). Using the *Clinical Evaluation of Language Fundamentals-Revised* (*CELF-R*), volunteer children had better receptive than expressive language skills, but referred children were depressed in both. Non-referred volunteer children had scores equivalent to national norms. Similarly, Greene, Kersey, and Prutsman (1973) found that *Seminole* school children, who live in the Florida Everglades, scored over one standard deviation below the norm on the *Wide Range Achievement Test* (*WRAT*), and this difference widened with age. Especially with children, while the *WISC-R* has good internal validity, it is also apparent that Native children are at a distinct normative disadvantage with commonly used *IQ* tests. Therefore, identifying *MMR* and *LD* Native children should be approached with caution, using local norms wherever possible, or by interpreting *IQ* scores and subtest profiles with extreme care. These findings also attested to the aggravating effects poverty contributes to the cultural equation.

Placement of Aboriginal children in foster homes is a contentious and even explosive issue, given the tragic history of residential schools. Cundick, Gottfredson, and Willson (1974) found that Native children enrolled at least five years in such an *Indian Education Programme* had educational achievement scores (i.e., on the *Iowa Test of Basic Skills* and *California Test of Mental Maturity*) that declined with successive years of participation. Moreover, these fostered children did no better in these residential schools than did those who remained at home and were educated in regular public schools. Also, their *IQ* scores did not

improve. Ironically, it is now known that *IQ* is largely a product of formal education (Ceci, 1991). Therefore, "rescuing" Aboriginal children from their reserves by placing them in foster care has no beneficial effects on their academic development. Notwithstanding the emotional abuse of taking children from their birth families, these results mirrored those of Garber and Begab (1988) in that, unless intervention occurs during the infancy and preschool periods, it does not reverse progressive intellectual decline. Moreover, successful interventions enter into therapeutic partnerships with parents.

Fetal Alcohol Syndrome, poverty and stress

Unfortunately, disentangling cultural differences from brain damage associated with drug and alcohol abuse, especially during pregnancy, is a difficult problem as poverty is a huge mediating factor. Vulnerability to *FAS*/*FAE* and lack of knowledge of *FAS* are positively related. For example, Binns (2001) found that primary and preschool teachers of *Alaskan* Native ethnicity scored significantly lower on knowledge and awareness of *FAS*. To be fair, it should be stressed that Native norms for drinking vary widely by community and subgroups within each community (May, 1991). Generally speaking, integrating *FAS*/*FAE* education within a supportive and receptive Native community is the obvious goal. As is reported below, *FAS* is at alarming levels within many Aboriginal communities and poverty potentiates the effects of alcohol on the fetus.

Drinking alcohol during pregnancy has become one of the largest sources of preventable contributors to mental retardation and behaviour disorders. Olofsson (2000) claimed that *FAS* is the most frequent cause of *MR* in Denmark and Niccols (1994) cites *FAS* as the most commonly known cause of *MR* in America. Phelps and Grabowski (1992) reported that *FAS* exceeds Down syndrome (*DS*), cerebral palsy (*CP*), and spina bifida (*SB*) as a cause of *MR*. Conry (1990) reported that, in rural and Native communities where maternal alcohol abuse and poverty are known concerns, prevalence rates of FAS/FAE are estimated as being about 190/1000 children, instead of the normative 0.4 to 3.1/1000. Astoundingly, that's approximately 112 times the norm. *FAE* is a milder

manifestation of the disorder in which facial dimorphisms may not be in evidence and the infant or child may lack one of three cardinal behavioural features, such a *MR*, inattentiveness or hyperactivity. Kavale and Karge's (1986) review showed that alcohol consumption during pregnancy was six times higher in lower-class than middle-class mothers, so poverty interacts with ethnicity and genetics. Niccols reported that during the first 4-10 weeks after conception, ethanol is a cytotoxin, which causes excessive cell death in the central nervous system (*CNS*) and abnormal nerve cell migration, leading to disorganized tissue structure. Early identification is essential, as recurrence in additional siblings is 77% (Abel, 1998). In a Scottish study, Plant (1984) controlled for age, smoking and legal and illegal drug use, finding that there was a significant correlation between self-reported maternal alcohol use and previous experiences of pregnancy termination or having a physically or mentally handicapped child or having a spontaneous abortion.

The *CNS* damage to the fetus as a result of *FAS* results in varying degrees of mental retardation, as a function of dosage, timing, duration and the uterine, placental, and health status of the particular mother. Holzman (1982) believes that damage sustained in the first trimester is more telling, although there is no known safe period during gestation. In addition to *MR*, such infants tend to have delayed growth, short eyelid slits, abnormal jaw protrusion, altered palmar crease patterns, wide-spaced eyes, a low forehead, joint and cardiac anomalies, and fine motor dysfunction (Furey, 1982). As Streissguth (1983) noted, such infants are often tremulous, irritable, and hypersensitive to sound, and have a weak sucking response. Streissgruth, La Due, and Schroeder (1987) described facial and cranial abnormalities in *FAS* children, such as microcephaly, short palpebral fissures (i.e., open eye width), flat midface, indistinct philtrum (i.e., upper lip), thin upper lip, epicanthal folds, low nasal bridge, minor ear abnormalities, short nose and micrognathia (i.e., small chin).

Using the *Developmental Behavior Checklist*, Steinhausen, Willms, Metzke, and Spohr (2003) found that, compared to controls (n = 15), infants, children and younger adolescents afflicted with *FAS* (n = 12) and *FAE* (n

= 26) were more disruptive, self-absorbed, anxious, antisocial and communication disordered. Hence, diagnostic inaccuracies may exist in identifying children with *ADD*-type hyperkinesis. Spatial memory may be a particular problem with *FAS* children (Uecker & Nadel, 1998), although Kaemingk and Halverson (2000) found that such deficits were due more to deficiencies in perceptual and verbal memory. Long-term outcomes for such children are not optimistic and include psychopathology, hyperkinesis, emotional disorders, sleep disorders, and abnormal habits and stereotypes (Steinhausen, Willms, & Spohr, 1993). Even eye problems, such as optic nerve hypoplasia and retinal dystrophy and reduced visual acuity, have been identified with this population (Stroemland, 1990). These findings also underscored the fact that, as with *ADHD*, *MMR*, and neglect, hyperkinesis is a major characteristic of these children. In an interesting comparison, Nanson (1990) found that *FAS* children, by middle childhood, are similarly affected by inattention and behaviour problems as are *ADD* children, apart from any intellectual decline.

While the direct teratogenic ethanol effects on the developing *CNS* are well known, specific teratogenic mechanisms are still being discovered. Ethanol administered to pregnant rhesus and cynomolgus monkeys has been found to interrupt feto-placental circulation, producing severe anoxia (Mukherjee & Hodgen, 1982). West, Hodges, and Black (1981) also found that ethanol changes the hippocampal mossy fibers in rats. Tsuji, Guizzetti, and Costa (2003) suspected that microencephaly associated with *FAS* is associated with ethanol-induced interrupted glial cell proliferation. These investigators found a dose-dependent decrease in phosphoproteins (i.e., MAPK and p70S6) necessary for neonatal cell proliferation. Stoltenburg-Didginger, and Spohr (1983) found that, in rats prenatally exposed to alcohol, the parietal spine distribution of proximal apical dendrites of layer V pyramidal cells were abnormal at 12 and 40 days postnatal, resulting in long, thin and entangled spines and a decreased number of normal stubby and mushroom-shaped spines.

There are many ways to get to Rome (i.e., *equifinality*) and so it seems to be the case with *FAS* and cell injury, as these above findings resembled those of normal karotype *MR* children. Pupura (1975) identified lower

dendtritic differentiation in *MR* children. Olney, Wozniak, Jevtovik-Todorovic, and Ikonomidou (2001) claimed that ethanol has the potential to create widespread apoptosis (i.e., cell death) during the synaptogenesis period of gestation. One intriguing explanation for the stronger link between *FAS*/*FAE* and poverty is the stress hypothesis. *GABA*-inducers, such as alcohol, help reduce stress and anxiety. Two processes, *NMDA* glutamate receptor blockade and excessive GABA-sub (A) receptor activation cause this cell death. Glutamate is the most common neuro-transmitter. Intriguingly, cell death is not confined to the gestation period, as Bellinger, Davidson, Bedi, and Wilce (2002) found that neonatal rat pups had significant glutamate changes due to daily prenatal alcohol exposure, resulting in decreased brain and body weight and microencephaly. Glial cells and synapses normally proliferate during the postnatal period.

Of course, in practice, it is difficult to identify the direct teratogenic effects of alcohol on the human fetus, as it is often the case that pregnant mothers are abusing other illicit substances as well and are often impoverished. To underscore the potential complexity of the diagnosis, Nanson (1992) found six cases where children were comorbid with *FAS* and autism. Asher (2002) found that cocaine may result in congenital abnormalities as well, such as stillbirth and intrauterine growth retardation. Heroin reduces fetal growth and increases the rate of fetal death. Likewise, even small amounts of alcohol can produce developmental delay and intellectual impairment (i.e., *FAE*) but with fewer and more minor dysmorphic features, compared with *FAS* associated with heavy drinking. Asher (2002) reported that dosage and duration are important, especially during organogenesis. This is the embryonic period from 18–60 days after conception, during which maximum cell damage is realized. During the fetal stage, growth is affected, especially to the brain.

Low birth weight. Low birth weight is the most consistent finding of alcohol-related birth defects (Sokol & Abel, 1992). Since heavy drinking is also associated with nutritional deficiencies (Able, 1980), particularly regarding niacin, mothers who are heavy drinkers are also more apt to deliver smaller babies (Landesman-Dwyer, 1982). Low birth weight is a

significant developmental risk. For example, Anderson and Doyle (2003) examined 568 8-year-old children who weighed 1,000 g or less at birth. Significantly lower *IQ*s were noted, as was the tendency to repeat a grade, to receive educational assistance, and to have an *LD*. Moreover, parents and teachers rated these children as less adaptive.

Nonspecific environmental factors and poverty. There is a complicated etiology of *MR* in vulnerable children (Abel, 1980; 1998; Neugut, 1982). For example, Taylor and Hall (1979) described the profile of mothers with previous stillborns who had inadequate opportunities for mourning or supportive help. Such mothers had unwanted babies, a lack of hospital rooming, were pregnant as teens, had emotional illnesses and were dependent on their parents. They also had lengthy separations from their newborns, lacked a husband's support, had a lower educational level and an iron deficiency. These impoverished mothers had a significantly more likely chance of having premature or clinically small babies requiring perinatal care. These authors also found that the most severe effect of alcohol on the fetus was spontaneous abortion. On the other hand, Ramey and Gowen (1986) found that systemic and intense intervention helps to prevent mild *MR* if administered before the second year, as indeed Garber and Begab (1988) found.

Genetics and biology. There are also quite demonstrable genetic vulnerabilities involved in alcohol exposure, as fraternal vs. identical twins are not likely to have comparably adverse outcomes. In addition, people of color or Black Americans are seven times more vulnerable to alcohol-related damage to their babies. The need for special after-care is especially important in such high risk births, as timely and continued support is essential in helping to prevent *MR* even in non-*FAS*/*FAE* infants. As well, the later-borns of alcoholic mothers are more severely affected, which is possibly related to greater blood alcohol concentrations as a result of liver decline and lower kidney clearance.

Of course, the literature is still largely silent on the confounding factors of early childhood poverty and deprivation (e.g., Garber & Begab, 1988) coupled with maternal alcohol use during pregnancy. Although Jones and Smith (1978) discovered *FAS* even in mothers controlling for

classical risk factors such as smoking, socio-economic class (*SES*) is nonetheless an essential consideration. For instance, Bingol, Schuster, Fuchs, and Iosub (1987) revealed that lower *SES* chronically alcoholic mothers (*n* = 48) were substantially more likely (71.9%) to have hyperactive offspring than were chronically alcoholic upper middle-class mothers (*n* = 36, 4.5%). Moreover, notwithstanding alcohol usage, hyperactivity in the offspring was also more likely (71%) in the lower *SES* mothers than in the upper middle-class mothers (21%). Congenital malformation, failure to thrive and *MR* were also higher in the lower *SES* group. The higher-class women ate regularly, had more balanced diets, and supplemented with vitamins and minerals. Whether these *SES* findings were due to differential stress levels, or nutritional differences, remains to be seen but at some basic level poor nutrition and stress are related.

It remains to be determined whether genetic selection, chronic stress, or the protective and buffering effects of antioxidants and other factors, such as nutrient malabsorbtion, differentiate socio-economically more vulnerable women from this serious *FAS* problem. Sapolsky's (2005) analysis of the poverty and health equation persuasively supports the stress hypothesis. Indeed, there seems to be good evidence for this. Chen, Langer, Raphaelson, and Matthews (2004) administered ambiguous (e.g., browsing in a store with an attentive saleswoman watching) and negative (e.g., other students teasing you) videos to high school students. Those students from lower *SES* backgrounds were significantly more likely to perceive threat during both the ambiguous and negative videos and had greater diastolic blood pressure and heart rate reactivity. According to Taylor, Branch, Van Zuylen, and Redei (1988), stress elevates corticosteroids, which when chronic and excessive, damage brain cells and produce similar behavioral problems as with *FAS*. The location of such damage is more pronounced in the hippocampus, the gateway to verbal memory (Feldman et al., 1997). Paradoxically, Taylor et al. argued that fetal alcohol exposure paradoxically activates stress hormones, which may actually aggravate the effects of alcohol in the economically deprived and chronically stressed population. Perhaps, inter-generational poverty makes mothers

more vulnerable to stress and alcohol abuse and in turn to having *FAS* offspring.

Interventions. Working with *FAE* children is very challenging, especially given the pernicious and pervasive nature of this disorder. However, Coe, Sidders, Riley, Waltermire and Hagerman (2001) have had some success using a variety of psychotropic interventions, such as stimulants, selective serotonin re-uptake inhibitors (*SSRI*s), mood stabilizers and neuroleptics. Aggressive treatment was advocated. If treatment is very difficult, then prevention is equivalently disconcerting. Murphy-Brennan and Oei (1999) found that, while *FAS* prevention programmes were good at raising awareness training, such programmes did not translate into lower alcohol consumption in the high risk, high consumption, group. Clearly, the prevention of adverse levels of poverty is crucial in lowering the incidence of *FAS*/*FAE*.

Conclusions

The deleterious effects of stressful levels of poverty on early child development are no longer subject to serious debate. Diminished development of verbal abilities and behavioral controls stems directly from maternal *MMR* and its effects on parenting and mother's lifestyle. Hyperactivity, inattentiveness and conduct problems otherwise attributed to well-defined developmental learning disorders may also stem from poverty. Unfortunately, due to exclusionary rules and administrative criteria, specific learning disorders are seldom applied to children with intellectual delays. Moreover, the effects of poverty are most common in the M*MR* range. Accordingly, criteria for funds for special educational services for those with *MR* are also not met. Consequently, standard diagnoses of learning disabilities and disorders such as *LDs* and *ADHD* may be under-identified for these children and adolescents and their learning needs fall through the proverbial cracks. Only through a careful history taking exercise using collateral sources is the practitioner or educator more likely to identify chronic poverty in the early years.

Poverty and ethnicity are also important in addressing the needs of poor children. Not only are new immigrants more likely to be under serious economic strain, but also the effects of early poverty must surely conspire to produce acculturation stresses. The incidence of *FAS*/*FAE* is much higher in the poor and aboriginal populations and it is behaviors associated with these conditions, such as hyperactivity, inattentiveness, poor memory, and slow learning, that may be confounded with other developmental diagnoses such *LD*s or *ADHDs*. Unless more accurate diagnoses are made, then interventions such as stimulant medications may be misapplied. Perhaps most importantly for the sake of the poor, current developmental diagnostic assessments tests of learning disorders need to be recalibrated to take into account these specialized populations. In this way, children from impoverished homes and lower *IQs* may not then be denied the specialized interventions that otherwise would be their right.

Is it too optimistic to propose that research efforts be conduced which target underprivileged children so that we may identify possible means to correcting the effects of early poverty and neglect? At the current time, the literature paints a very gloomy picture for such children when interventions are postponed until after school entry. Surely, revitalizing neurological development in young children is not beyond our science at this point? In the meantime, the war on poverty is a real one and deserves our dedicated effort.

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